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Cardiometabolic Profiles of Adolescents and Young Adults Exposed to the World Trade Center Disaster

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Abstract

Background and Objective—Few studies have examined the possible cardiometabolic consequences of World Trade Center-related exposures on children who lived and/or attended school near the disaster site. Our objective was to compare cardiometabolic profiles of participants in the World Trade Center Health Registry (WTCHR) with a matched comparison group.

Methods—We evaluated WTCHR enrollees who resided in New York City and were born between September 11, 1993 and September 10, 2001, and a matched comparison group. We

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Institutional Review Board (IRB) Approval

The study was reviewed and approved by the NYU School of Medicine IRB and research committees at Bellevue and Gouverneur Hospital Centers. The NYC Department of Health & Mental Hygiene (NYC DOHMH) Institutional Review Board identified this study not to involve human subject activity by NYC DOHMH staff. Adolescents <18 years of age provided informed assent forms along with parental informed consent forms before undergoing study procedures. A Certificate of Confidentiality was obtained to protect participant privacy.

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assessed exposure to dust cloud, home dust, as well as traumatic exposure, and associations with blood pressure, arterial wall stiffness, body mass index (BMI), total cholesterol, triglycerides, HDL, and LDL.

Results—A total of 402 participants completed the study, 222 in the comparison group and 180 in the WTCHR group. In multivariable regression analysis, after adjusting for relevant confounders we detected a weak association between participation in the WTCHR group and lower BMI (−1.12 kg/m^2, 95% CI −2.11, −0.12; p=0.03), which became non-significant after adjusting for multiple comparisons. With respect to traumatic and psychosocial exposures, the only association that persisted in our multivariable model, below our predefined level of significance, was between post-traumatic stress disorder and higher BMI (2.06 kg/m2, 95% CI 0.37, 3.74; p=0.02).

Conclusions—Our findings do not support an association between self-reported exposures to the WTC disaster and adverse cardiometabolic profile. However, further longitudinal studies may better inform the full extent of WTC-related conditions associated with exposure to the disaster.

Keywords

World Trade Center Disaster; cardiometabolic effects; dust exposure; traumatic exposure

1. Introduction[a]

Physical health consequences of the World Trade Center (WTC) disaster are well documented for rescue and recovery workers, adult residents (Crowley et al., 2011; Dalton et al., 2010; Izbicki et al., 2007; Prezant et al., 2002; Reibman et al., 2005; Reibman et al., 2009), and perinatally-exposed children (Berkowitz et al., 2003; Lederman et al., 2004; Wolff et al., 2005), yet data are extremely limited about physical health impacts on the thousands of children who lived and/or attended school below 14th Street in downtown Manhattan on September 11, 2001 (Landrigan et al., 2004). In addition to acute dust cloud exposures, local residents experienced significant subchronic exposures from fires and resuspended dust, which entered homes and schools through windows and air shafts (Friedman et al., 2011a).

Existing studies have documented increases in asthma prevalence (Stellman et al., 2013) and decrements in pulmonary function (Trasande et al., 2013) in children exposed to the 9/11 disaster, but few studies have examined the possible cardiometabolic consequences of WTC-related exposures in early life. Indeed, especially for those children with asthma and other respiratory conditions associated with exposure to the WTC disaster, obesity, insulin resistance and other cardiometabolic disorders may well exist. Increased risk of obesity among children with asthma is well documented, possibly due to decreased functional lung and exercise capacity (Shore, 2008). In addition, dietary imbalances may result from mental health consequences, such as depression, anxiety and post-traumatic stress (Goodman and

[a]**Abbreviations:** Body Mass Index (BMI); Generalized anxiety disorder (GAD); High-density lipoprotein (HDL); Low-density lipoprotein (LDL); Major depressive disorder (MDD); New York State Department of Health (NYSDOH); NYC Department of Health & Mental Hygiene (NYC DOHMH); Posttraumatic stress disorder (PTSD); World Trade Center (WTC); WTC Health Registry (WTCHR).

Whitaker, 2002) that developed following exposure to WTC dust or witnessing the events of September 11, 2011.

In adults, previous studies have shown that markers of metabolic syndrome such as abnormal triglycerides and HDL are independent risk factors of greater susceptibility to lung function impairment after September 11, 2001 (Naveed et al., 2012). In addition, cardiovascular biomarkers in serum six months after September 11, 2001 predicted changes in lung function in firefighters exposed to dust from the WTC (Weiden et al., 2013). Previously published data from the WTC Environmental Health Center/Survivors Health Program suggested a high rate of pre-hypertension (45.5% across all those ≤18 years old on September 11, 2001, 33.3% among those <20 years of age at the time of assessment) as well as reductions in HDL and elevations in triglycerides in a subset of children with chronic residual home dust exposure following the disaster (Trasande et al., 2013). However, there are limits in comparing results obtained from clinically studied samples. Without a proper comparison group, other environmental exposures and lifestyle choices that have persisted in the 15 years since the disaster could represent an alternative explanation to the possible cardiometabolic alterations present in exposed children. The World Trade Center Health Registry (WTCHR) is a registry sponsored by the National Institute for Occupational Safety and Health, which follows the most extensive sampling of adolescents potentially exposed to the disaster (Farfel et al., 2008). On the basis of this preliminary data, we hypothesized that children who were exposed to the WTC disaster (exposure to dust cloud, home dust or traumatic exposures) would have adverse cardiometabolic profile, as measured by arterial wall stiffness, body mass index, fasting total cholesterol, HDL, LDL and triglycerides. To investigate this hypothesis, we aimed to examine cardiometabolic profiles of WTCHR participants and a matched comparison group, to investigate potential differences between the two groups related to exposure to the WTC disaster.

2. Participants and Methods

2.1 Study Participants

In order to maximize contrasts between the two study populations and differentiate effects that may be due to the disaster, we recruited (1) a cohort of New York City residents enrolled in the WTCHR with dates of birth on or between September 11, 1993 and September 10, 2001 and (2) a comparison cohort of individuals born during the same time period, who were ineligible for enrollment in the WTCHR because they either did not reside south of Canal Street, did not attend school south of Canal Street, and were not present south of Chambers Street on the morning of 9/11(Friedman et al., 2011b). Nonetheless, we recognized that WTC exposures were widespread in the aftermath of the disaster, and so we also queried participants and their family members about relevant exposures (see Exposures and Covariates below).

To enroll members of the WTCHR cohort in the present study, WTCHR staff of the New York City Department of Health (NYCDOHMH) who were fluent in English, Spanish, Mandarin, or Cantonese attempted contacts by mail, email, phone and in-person visits. Both a hard-copy letter and brochure describing the study were mailed to each potential participant. Two weeks after the mailing, phone calls were initiated to individuals who had

not responded to the mailed invitation to participate. Calls were made to all known telephone numbers, and calls were attempted at different hours of the day and evening, and on different days of the week. Emails were sent to potential participants who did not respond to mail or telephone contacts. If there was no response to emails, then Lexis-Nexis (RELX Group: New York City, NY) search tools were used to identify new contact information. If new contact information was identified, then telephone and/or email contact were reinitiated. If no new contact information was obtained from tracing, two WTCHR staff members attempted a home visit to the last known place of residence. In all methods of contact, WTCHR staff described the study and invited individuals to call the WTCHR or NYU School of Medicine staff to further discuss study details and make an appointment. For participants less than 18 years of age a parent or guardian was required to schedule an appointment and be available and present to authorize participation on the scheduled visit date.

For our comparison group, we recruited individuals who were not eligible for enrollment in the WTCHR. To maximize comparability between the two groups of study participants we developed a table of the desired frequencies of controls by age (0–2, 3–5 or 6–8 years-old on 9/11/2001, with age 8 years being the upper bound for age restriction), sex, race (White, African-American, Asian, other), ethnicity (Hispanic, non-Hispanic) and income (<$25,000, ≥$25,000), assuming that the enrolled group of WTCHR participants would reflect participants in the WTCHR's most recent (2011–12) survey cycle. Three modes of recruitment were employed to recruit the frequency-matched comparison group: (1) well visits at pediatric clinics affiliated with NYU School of Medicine; (2) contact through health fairs, youth organizations, and postings in areas where youth congregate, posting and advertisements at local colleges; and (3) social media outreach by West Coast Clinical Trials (WCCT) Global, a contract research organization. Participants' eligibility and ability to fill slots in the frequency-matching table were assessed using a screening questionnaire, which staff conducted over the phone or in person. Individuals were excluded from the present study as matched comparisons if they would have qualified for WTCHR enrollment because of place of residence or school, or having been in the vicinity of the WTC towers on 9/11/2001.

Exclusion criteria—Participants were not considered eligible for either the WTCHR or the control group if any of the following was present: i) inability to follow study procedures for measurement of arterial stiffness; ii) serious lung or heart condition (severe cardiac or pulmonary congenital anomalies; iii) heart or lung surgery; iv) pregnancy.

In addition, for participants with any current illnesses within two weeks of the study visit such as the common cold, pneumonia, asthma exacerbation, a two-week hiatus was given for these current illnesses to resolve. Participants with controlled asthma were not excluded.

2.2 Study visits

Visits took place on evenings, weekends and during school holidays to maximize convenience either in 1 or 2 visits at the study site. After providing informed consent, the following were performed: a fasting blood draw; anthropometric measurements; and blood

pressure/brachial artery distensibility/pulse wave velocity measurements. Study visits took place in an NYU Medical Center facility from February 20, 2014 to March 21, 2016.

Fasting Blood Draw—Participants were instructed to fast for six hours before study visits, and to avoid food, caffeine-containing products, and sugary drinks. We measured fasting total cholesterol, triglycerides, HDL, and LDL (calculated).

Assessment of Adiposity—Weight and height were measured using calibrated stadiometers (Shorr Productions, Olney, MD) and scales (Seca model 881; Seca Corp., Hanover, MD). Body Mass Index (BMI) Z-scores were derived from 2000 Centers for Disease Control and Prevention (CDC) norms, incorporating height, weight and sex; overweight and obese were categorized as BMI Z-score ≥1.036 and ≥1.64 (Ogden et al., 2002). Since some of the participants were older than 20 years at the time of the study visits, we also included BMI in kg/m^2 in our analysis.

Brachial Artery Distensibility (BrachD)—Increases in blood pressure (BP) are late signals of vascular dysfunction, and do not directly measure changes in the arterial wall, where we posit pathophysiologic effects related to WTC exposures may occur. Brachial artery distensibility (BrachD) measurement is a rapid method of accurately assessing the relative stiffness of a peripheral artery and may detect early vascular alterations that would not be detected by simple BP measurement. The DynaPulse Pathway instrument derives BrachD and BP using the technique of pulse waveform analysis of arterial pressure signals obtained from a standard cuff sphygmomanometer. A lower value of BrachD indicates a stiffer vessel (Urbina et al., 2002). Following a 5 minute rest period**,** a BP cuff appropriate for the subject's upper arm size was applied, and three automatic recordings of systolic, diastolic, mean arterial BP and heart rate were obtained. Off-line analyses of brachial artery pressure curve data were then performed by Pulse Metric, Inc. using an automated system to derive parameters from the pulse curves to calculate BrachD (Urbina et al., 2011). We followed the common practice of averaging BP measurements for purposes of generating continuous and categorical BP variables. Because BP varies widely by age, sex and height, we calculated systolic/diastolic BP Z-scores from mixed-effects linear regression models derived using data from 1999–2000 National Health and Nutrition Examination Survey (National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents, 2004).

Central Arterial Wall Stiffness Assessment—Compared with BP and brachial artery distensibility assessments, pulse wave velocity (PWV) is the most sensitive measure of arterial wall stiffness, reflecting the speed for the pressure wave generated by cardiac ejection to reach the periphery. A higher value indicates a stiffer vessel. PWV is considered extremely useful to detect early signs of arterial stiffness in children (Urbina et al., 2009), and increased PWV has been identified in obese and type 2 diabetic children (Urbina, 2010).

PWV was measured by obtaining the arterial pulse waveform at the common carotid and femoral arteries using the SphygmoCor CPV System (AtCor Medical, Sydney, Australia) (Laurent et al., 2006). Arterial waveforms gated to the R-wave on the ECG tracing are recorded from the carotid then distal artery of interest, and PWV is then calculated as the

difference in the carotid-to-distal path length divided by the difference in R-wave-to-waveform foot times. Recent data have shown excellent reproducibility with coefficients of variability < 7% even in obese adolescents (Urbina, 2010). The SphygmoCor CPV System was also used to measure central aortic pressure and the Augmentation Index (AIx), a vascular parameter incorporating both central stiffness and wave reflections, with a higher value indicating arterial dysfunction (McEniery et al., 2006).

2.3 Exposure and covariates

Dust exposures experienced by children living and attending school near the WTC site—Acute exposure to dust cloud was determined as present/absent with the question: “Were you caught in the WTC dust or debris cloud in the morning after the buildings collapsed on 9/11?” Sub-chronic and chronic exposure to home dust was determined present/absent with the question: “In the year after 9/11/01, did you live in an apartment or home in which WTC dust was visible on surfaces at any time, even if only briefly?” An affirmative response given by either the adolescent or the parent/guardian indicated exposure.

Psychologically Stressful Exposures Experienced by Children Living and Attending School Near the WTC Site—Psychologically traumatic exposures in both groups were measured with the eight-item questionnaire developed by Comer and colleagues (Comer et al., 2010). Both study participants and parents/guardians were administered these questionnaires. Parents/guardians answered about their own exposures as well as their children’s exposure, and answers were then compared. Parent trauma was not used in the analysis. Traumatic exposure was noted if either the adolescent or parent/guardian gave a positive response to the questionnaire items of: sight of either tower collapse, sight of injured people, sight of dead bodies, sight of people falling out of buildings, physical injury to self, need to depart home/work for safety, and worry about safety of a loved one.

We also measured exposure to trauma other than the WTC attacks since previous evidence suggests that exposure to the WTC disaster and to other trauma is synergistically associated with clinically significant behavioral problems among young children (Chemtob et al., 2008). For this purpose, we used the UCLA Child PTSD-Reaction Index (UCLA-RI), which consists of a brief trauma checklist that enquires about lifetime exposure to a wide variety of events, such as community violence, medical trauma, abuse, and domestic violence (Steinberg et al., 2004). Both adolescent and parent were asked to complete the UCLA-RI trauma checklist. The parent reported his/her own trauma history as well as the trauma history of his/her child.

If an adolescent participant screened positive for any trauma exposure, including WTC exposure, post-traumatic stress disorder (PTSD) symptoms and their frequency were then assessed with the same instrument used to assess exposure to other lifetime trauma (UCLA-RI). A sum of symptom item scores was used to index PTSD symptom severity, and identify present/absent PTSD using validated criteria (Pynoos et al., 1998). If a parent reported trauma exposure of his/her child, the parent was asked to complete the UCLA-RI to report

his/her child's symptoms and frequency. Subjects with a score of 10 or higher were considered to have clinically significant PTSD symptoms (Cohen et al., 2008).

Additional Covariates—To obtain dietary data, participants completed a web-based version of the Diet History Questionnaire II (DHQ II), a publicly available food frequency questionnaire (FFQ) developed by the National Cancer Institute. Using the DHQ II, we collected data regarding food consumption in the past month with portion size information, to ensure accuracy in caloric intake data. The DHQ II measure has been validated in previous studies as providing equal or improved nutritional information to the Willett and Block FFQs instruments (Rockett et al., 1997). Participants also completed a three-day physical activity diary, based on the International Physical Activity Questionnaire-Short Last Seven Days, which is well validated (Craig et al., 2003). Physical activity data from the diary were converted into energy expenditure estimates as MET using published values (Ainsworth et al., 2000).

Other identified covariates included BMI category (normal/overweight/obese), race/ethnicity (White, African American, Asian, Other and Hispanic), sex, and exposure to tobacco smoke. This was evaluated by saliva cotinine concentration and questionnaire. We analyzed salivary cotinine using a highly sensitive (limit of detection 0.15 ng/mL) test from Salimetrics, Inc. (State College, PA). Cotinine was categorized into low (<0.15 ng/mL), medium (≥0.15 to < 2.32 ng/mL), and high (≥2.32 ng/mL) (Bernert et al., 2000). For subjects without saliva cotinine concentration, we categorized using questionnaires (low: no smoker and no secondhand smoke exposure; medium: no smoker but secondhand smoke exposure; high: smoker).

2.4 Sensitivity analyses

We assessed the robustness of our analysis by reprising (1) multivariable analyses including only subjects with cotinine data as a categorical variable and adjusting for age as a continuous variable and (2) multivariable analyses including only subjects with cotinine data and excluding subjects with any kind of WTC exposures (home dust, dust cloud, and traumatic exposures) in the comparison group.

2.5 Institutional Review Board (IRB) Approval

The study was reviewed and approved by the NYU School of Medicine IRB and research committees at Bellevue and Gouverneur Hospital Centers. The NYC Department of Health & Mental Hygiene (NYC DOHMH) Institutional Review Board identified this study not to involve human subject activity by NYC DOHMH staff. Adolescents <18 years of age provided informed assent forms along with parental informed consent forms before undergoing study procedures. A Certificate of Confidentiality was obtained to protect participant privacy.

2.6 Statistical Analysis

We conducted descriptive, univariate, and multivariate analyses with R Statistical Software (version 3.3.1). Chi-square test or Fisher exact test was used to compare the sociodemographic and exposure variables of each cohort. Wilcoxon Rank Sum test was used

to compare caloric intake, physical activity levels, and cardiometabolic markers. BMI, triglycerides and cholesterol levels were log-transformed to account for skewed distribution. Chi-square analyses also compared sociodemographic variables between WTCHR adolescents and young adults who were recruited for the present study and other WTCHR adolescents and young adults who did not participate.

Correlation coefficients were calculated for the three exposure variables. To avoid multicollinearity, separate multivariate models examined study group; dust cloud; home dust; and traumatic exposures. Univariate linear and logistic regression was used to compare outcomes by traumatic and psychosocial factors. Multivariable) linear regression or multiple logistic regression was used for continuous or discrete outcomes, controlling for sex, race/ethnicity, caloric intake, physical activity, smoke exposure, BMI category (the latter not included if BMI related outcomes). Variables were added to multivariate models if significant differences were identified between the two comparison groups at $p<0.1$. All statistical tests were two-sided and, in recognition of the four simultaneous comparisons and the need for Bonferroni correction (Bonferroni, 1935), we used a significance cutoff of 0.01 (~0.05/4) for the univariate and multivariate regression analyses

3. Results

Figure 1 provides an overview of the enrollment process for the WTCHR study participants and matched comparison group. Compared to other WTCHR enrollees, those who took part in the present study were older (P=0.004) and more likely to have higher family income (P<0.001; Appendix Table 1, Supplemental Information).

Despite our substantial efforts to recruit unexposed comparisons (Table 1A, P<0.001), WTC exposures were also identified in the comparison group: 0.5% to dust cloud; 7.7% to home dust; and 42.8% for traumatic exposures. Participants in the comparison group were also more likely to be female (P= 0.008). The presence of PTSD was more likely in the WTCHR group (P=0.04), whereas major depressive disorder (MDD) was more likely to be present in matched comparisons (P=0.05). Caloric intake was higher in the WTCHR participants (p=0.03), and overweight and obesity were more likely in comparisons (P=0.05) yet overweight and obesity were not significantly different between cohorts among those who agreed to venous blood sampling (P=0.39, Table 1B). The number of participants who provided blood samples was 123 in the WTCHR group and 185 in the comparison group (Table 1B), and total cholesterol, triglycerides, HDL and LDL are reported only for these participants.

Self-reported exposures were correlated with each other supporting separate multivariable models (Appendix Table 2, Supplemental Information). Univariable analyses confirmed differences in key covariates supporting inclusion in multivariable models (Appendix Tables 3–5, Supplemental Information). In multivariable linear regressions adjusting for sex, race/ethnicity, caloric intake, physical activity, and exposure to tobacco smoke, we identified associations between participation in the WTCHR group with lower BMI (continuous,−1.12 kg/m^2, 95% CI −2.11, −0.12; P=0.03) and BMI z-score (continuous, −0.24, 95% CI −0.49, −0.002; P=0.05) (Table 2), and between PTSD (presence/absence) and higher BMI

(continuous, 2.06 kg/m^2, 95% CI 0.37, 3.74; P=0.02). Home dust exposure was associated with greater brachial artery distensibility (0.31, 95% CI 0.02, 0.60; P=0.04), after also adjusting for BMI. None of these associations, however, were significant after correction for multiple comparisons.

In logistic regressions (Table 3), after adjusting for the same key covariates, dust cloud (OR 1.12, 95% CI 1.04, 1.21; P=0.004) was significantly associated with increased odds of PTSD, as was belonging to the WTCHR group (OR 1.10, 95% CI 1.03, 1.16; P=0.002). There were other modest associations with PTSD, but these did not reach significance after correction for multiple comparisons.

In our sensitivity analyses, first we included only subjects with cotinine data. We had 85 subjects with missing cotinine data (71 in the comparison and 14 in the WTCHR group), leaving a sample size of 317. The associations between participation in the WTCHR group and lower BMI were no longer significant, but the association between PTSD (presence/absence) and higher BMI (continuous, 2.10 kg/m^2, 95% CI 0.38, 3.82; P=0.02), remained significant. While home dust exposure was no longer associated with greater brachial artery distensibility, PTSD resulted significantly associated with decreased brachial artery distensibility (−0.68, 95% CI −1.21, 0.15; P=0.01) (Appendix Table 6, Supplemental Information). In logistic regression, dust cloud exposure remained significantly associated with PTSD (OR 1.11, 95% CI 1.02, 1.20; P=0.02) (Appendix Table 7, Supplemental Information). When we adjusted for age as a continuous variable, results did not change (Appendix Tables 8 and 9, Supplemental Information).

When we reprised multivariable analyses including only subjects with cotinine data and excluding subjects with any kind of WTC exposures (home dust, dust cloud, and traumatic exposures) in the comparison group, the results were essentially unchanged, and the significant association between PTSD (presence/absence) and higher BMI persisted (Appendix Tables 10 and 11, Supplemental Information).

4. Discussion

This research study found that self-reported exposures to the WTC disaster were weakly associated with cardiometabolic profiles, and non-significant after correction for multiple comparisons. Among the weak associations detected, the relationship between PTSD and BMI is consistent with studies suggesting that PTSD is associated with unhealthy behaviors such as increased alcohol use, smoking, and increased caloric intake (Dedert et al., 2010). Furthermore, PTSD has been previously associated with higher blood pressure, BMI, and metabolic syndrome, even in comparison with other psychiatric disorders (Farr et al., 2015; Jin et al., 2009; Pagoto et al., 2012), as well as new-onset diabetes in a WTC-exposed cohort (Miller-Archie et al., 2014). Given that PTSD has been shown to be one of the most common mental health consequences in populations exposed to the WTC disaster, its associations with increased BMI in our analysis deserve further investigation and warrant possible inclusion of PTSD among the list of risk factors for the possible development of metabolic dysfunctions in this exposed population. In fact, if confirmed in further longitudinal studies in this population, such BMI increments are significant when considered

at the population level, in which even small increments can result in large increases in overweight and obesity prevalence, shifting the distribution of BMI and increasing the number of individuals who are above the cut off points for overweight and obesity.

We note substantial limitations to interpretation. Participation rate especially in the WTCHR group was modest, possibly due to reluctance of parents to have their children recall the disaster and due to the long time period that had lapsed since the disaster. Furthermore, this was a dynamic population with issues of relocation, school schedules/commitments and going away to college. Adolescents below the age of 18 were required to be accompanied by their parent/guardian and the time commitments for the study visits also represented an obstacle to participation for some. Recruitment of unexposed comparisons occurred at a higher rate though this too was modest. Both groups may have participation bias that could explain the lack of findings in some cases and the significant associations in others. For example, potential participants reporting both a major exposure like dust cloud and a major symptom like PTSD could have been more likely to take part in the study than those with only an exposure and no major symptom or symptoms but no exposure. This could lead to selection bias away from the null and could contribute to explaining an OR of 1.12 for the association between PTSD and exposure to dust cloud.

We also suffered from contamination of our unexposed group, even though we compared the groups and exposures across groups. It is also possible that effects are confounded by interceding exposures between the time of the disaster and the present study. While these are serious limitations, we emphasize the novelty and importance of this study, and arguably the lack of a superior alternative, with the exception of biomarkers of WTC contaminants, which are superior at this time frame insofar as they are related to known exposures as recalled, and described in our previous manuscript in which we documented differences in serum perfluoroalkyl acids both by group and exposure as reported (Trasande et al., 2017).

While some reassurance can be interpreted from this study insofar as most of the associations were nonsignificant, even for subclinical markers of peripheral and central arterial wall stiffness (brachial artery distensibility and PWV), longitudinal studies may also be more informative in assessing cardiometabolic effects of the disaster.

5. Conclusion

Our findings do not support an association between self-reported exposures to the WTC disaster and adverse cardiometabolic profile. However, we cannot exclude that assessment of biomarkers may reflect more accurately actual exposure status of this population. Further longitudinal study may better inform the full extent of WTC-related conditions associated with exposure to the disaster.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- WTC-related exposures may be associated with cardiometabolic consequences
- Cardiometabolic profiles of exposed youth were compared with a matched group
- Self-reported exposures were not associated with adverse cardiometabolic profile
- Longitudinal studies may be more informative in assessing WTC-related consequences

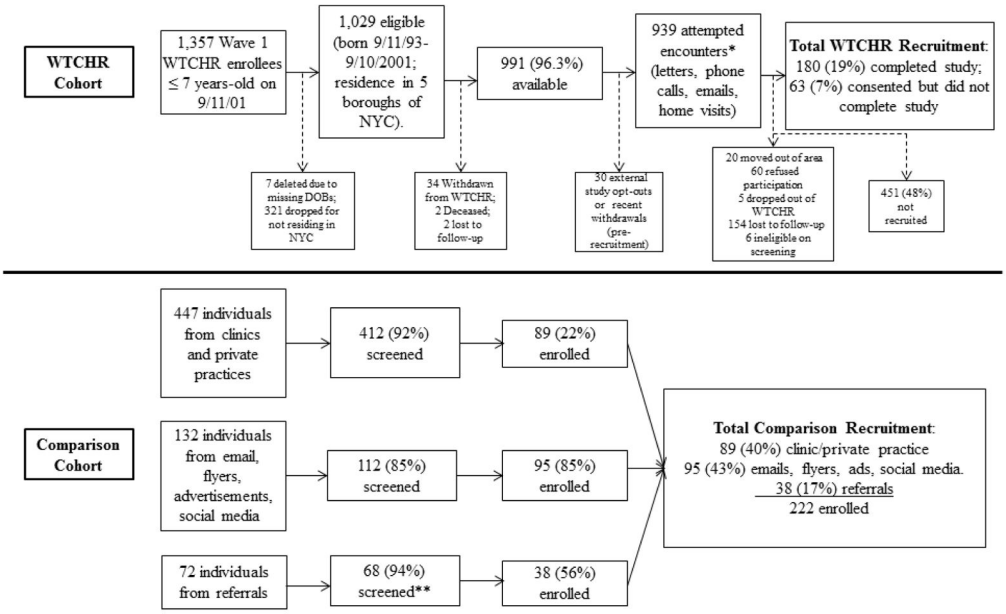

Figure 1. Recruitment Flowchart for WTCHR and Comparison Cohorts

*The number of attempted encounters (939) is the number of potential participants that our study team was able to approach

**A total of 592 subjects were screened: 222 were enrolled, 103 were considered ineligible, 96 refused participation, and 171 were considered lost to follow up

Table 1A

Characteristics of the two study populations.

	Control group n=222	WTCHR group n=180	p value
Sex			
Male	89 (40.1%)	97 (53.9%)	**0.008**
Female	133 (59.9%)	83 (46.1%)	
Date of birth			
9/11/93–9/10/95	45 (20.3%)	47 (26.1%)	0.159
9/11/95–9/10/98	89 (40.1%)	77 (42.8%)	
9/11/98–9/10/01	88 (39.6%)	56 (31.1%)	
Income < $25,000[a]	49 (27.4%)	28 (19.4%)	0.126
Race/Ethnicity[b]			
Non-Hispanic White (%)	89 (40.1%)	66 (36.9%)	0.053
Non-Hispanic Black (%)	19 (8.6%)	16 (8.9%)	
Non-Hispanic Asian (%)	44 (19.8%)	49 (27.4%)	
Non-Hispanic Other (%)	10 (4.5%)	16 (8.9%)	
Hispanic (%)	60 (27.0%)	32 (17.9%)	
Exposures, n (%)			
Dust cloud exposure	1 (0.5%)	61 (38.6%)	**<0.001**
Home dust exposure	17 (7.7%)	98 (56%)	**<0.001**
Traumatic exposure	95 (42.8%)	150 (83.3%)	**<0.001**
Caloric intake,[c] Median (IQR)	1535 (1061, 2087)	1621 (1141, 2331)	**0.028**
Physical activity, MET hours per week (IQR)	150 (90, 240)	180 (120, 285)	**0.087**
Body Mass Index Category			
Normal weight/underweight	162 (73.0%)	150 (83.3%)	**0.045**
Overweight	36 (16.2%)	19 (10.6%)	**0.028**
Obese	24 (10.8%)	11 (6.1%)	0.087
Smoking status			
Smokers	23 (10.4%)	24 (13.3%)	0.443
Median Cotinine Concentration	0.324 (0.106, 0.690)	0.412 (0.106, 0.984)	0.294
Tobacco smoke exposure[d]			
--Low (<0.15 ng/mL)	102 (45.9)	73 (40.6)	0.353
--Medium (≥0.15 to < 2.32 ng/mL)	95 (42.8)	79 (43.9)	0.443
--High(≥2.32 ng/mL)	25 (11.3)	28 (15.6)	0.294
Mental Health- Psychosocial exposures			
Post-Traumatic Stress Disorder	14 (6.3%)	23 (12.8%)	**0.04**
Major Depressive Disorder	55 (24.8%)	29 (16.1%)	**0.045**
General Anxiety Disorder	11 (5%)	6(3.3%)	0.58
Cardiometabolic Markers, Median (IQR)			
Triglycerides (mg/dL)	66.5 (48, 95.3)	63.5 (49.8, 88.5)	0.891
High-Density Lipoprotein (mg/dL)	53 (44, 66)	52 (43.75, 60.25)	0.294

	Control group n=222	WTCHR group n=180	p value
Low-Density Lipoprotein (mg/dL)	77 (66, 94)	80 (69, 96)	0.131
Total Cholesterol (mg/dL)	148.5 (133, 166.3)	148.5 (133, 170)	0.827

[a] n=43 missing for comparison group; n=27 missing for WTCHR group;

[b] n=1 missing for race/ethnicity;

[c] n=2 missing for caloric intake;

[d] Evaluated by saliva cotinine concentration and questionnaire.

For subjects without saliva cotinine concentration, we categorized no smoker and no secondhand smoke exposure into “low”, no smoker but secondhand smoke exposure into “medium”, and smoker into “high” category

Table 1B

Characteristics of the populations who provided fasting blood.

	Comparison (n=185)	WTCHR (n=123)	p value
Sex, n (%)			
Male	74 (40%)	69 (56.1%)	**0.010**
Female	111 (60%)	54 (44.9%)	
Date of birth, n (%)			
9/11/93–9/10/95	35 (18.9%)	34 (27.6%)	
9/11/95–9/10/98	73 (39.5%)	52 (42.3%)	0.070
9/11/98–9/10/01	77 (41.6%)	37 (30.1%)	
Income < $25,000[a]	42 (22.7%)	19 (15.4%)	0.170
Race/Ethnicity,[b] n (%)			
Non-Hispanic White	72 (38.9%)	42 (34.4%)	
Non-Hispanic Black	17 (9.2%)	13 (10.7%)	
Non-Hispanic Asian	37 (20%)	30 (24.6%)	**0.040**
Non-Hispanic Other	6 (3.2%)	13 (10.7%)	
Hispanic	53 (28.6%)	24 (19.7%)	
Exposures,[c] n (%)			
Dust cloud exposure	1 (0.5)	47 (43.9)	**<0.0001**
Home dust exposure	15 (8.1)	69 (57)	**<0.0001**
Traumatic exposure	80 (43.2)	109 (88.6)	**<0.0001**
Calories,[d] Median (IQR)	1537 (1014)	1709 (1317)	**0.008**
Tobacco smoke exposure			
Low	102 (45.9)	73 (40.6)	
Medium	95 (42.8)	79 (43.9)	0.353
High	25 (11.3)	28 (15.6)	
Body Mass Index Category			
Normal weight/underweight	137 (74.1)	98 (79.7)	
Obese	20 (16.3)	8 (4.3)	0.387
Overweight	28 (15.1)	17 (13.8)	

[a] n=38 missing for comparison; n=27 missing for WTCHR

[b] n=1 missing for race/ethnicity

[c] n=18 missing for dust cloud exposure; n=2 missing for home dust exposure

[d] n=2 missing for caloric intake

Table 2

Linear Regression Analysis of Cardiometabolic Outcomes Associated with Dust Cloud, Home Dust, belonging to WTCHR group, and traumatic and psychosocial exposures (N=402 subjects)

	Multivariable analysis					
	Dust cloud		**Home dust**		**WTCHR**	
Cardiometabolic Outcomes	***unit change (95% CI)***	***p value***	***unit change (95% CI)***	***p value***	***unit change (95% CI)***	***p value***
BMI (kg/m^2)	−1.12 (−2.50, 0.25)	0.11	−0.68 (−1.77, 0.40)	0.22	−1.12 (−2.11, −0.12)	**0.03** *
BMIz	−0.20 (−0.52, 0.11)	0.21	−0.16 (−0.41, 0.10)	0.24	−0.24 (−0.49, −0.002)	**0.05** *
logTrig	−0.05 (−0.18, 0.09)	0.50	−0.003 (−0.11, 0.10)	0.96	0.02 (−0.07, 0.12)	0.64
logChol	0.02 (−0.03, 0.07)	0.47	−0.01 (−0.05, 0.04)	0.78	0.02 (−0.02, 0.06)	0.33
logLDL	0.05 (−0.04, 0.13)	0.30	0 (−0.07, 0.07)	0.99	0.06 (−0.001, 0.12)	0.05
logHDL	0.02 (−0.07, 0.11)	0.62	−0.03 (−0.10, 0.04)	0.38	−0.04 (−0.10, 0.03)	0.26
BrachD	0.22 (−0.14, 0.58)	0.23	0.31 (0.02, 0.60)	**0.04** *	0.18 (−0.09, 0.45)	0.20
AIx	−0.24 (−3.19, 2.71)	0.88	0.25 (−2.08, 2.58)	0.83	−1.006 (−3.19, 1.17)	0.37
PWV	−0.08 (−0.28, 0.12)	0.43	−0.12 (−0.27, 0.04)	0.15	−0.01 (−0.24, 0.05)	0.18

	Other Traumatic Exposure		**PTSD**		**Major Depressive Disorder**		**General Anxiety Disorder**	
Cardiometabolic Outcomes	*unit change (95% CI)*	*p value*	*unit change (95% CI)*	*p value*	*unit change (95% CI)*	*p value*	*unit change (95% CI)*	*p value*
BMI (kg/m^2)	−0.14 (−1.15, 0.86)	0.78	2.06 (0.37, 3.74)	**0.02** *	−0.29 (−1.51, 0.92)	0.64	1 (−1.45, 3.45)	0.42
BMIz	−0.15 (−0.39, 0.09)	0.21	0.08 (−0.31, 0.46)	0.69	−0.18 (−0.47, 0.12)	0.25	−0.11 (−0.67, 0.45)	0.70
logTrig	−0.04 (−0.14, 0.05)	0.38	0.15 (−0.005, 0.31)	0.06	−0.05 (−0.16, 0.06)	0.41	−0.03 (−0.25, 0.18)	0.77
logChol	0.007 (−0.03, 0.05)	0.73	0.06 (−0.007, 0.12)	0.08	0.004 (−0.04, 0.05)	0.86	−0.05 (−0.14, 0.04)	0.26
logLDL	0.06 (−0.006, 0.12)	0.08	0.05 (−0.05, 0.15)	0.32	0.004 (−0.07, 0.08)	0.91	−0.02 (−0.16, 0.11)	0.73
logHDL	−0.06 (−0.12, 0.01)	0.10	0.07 (−0.04, 0.17)	0.22	0.03 (−0.05, 0.10)	0.49	−0.08 (−0.22, 0.07)	0.29
BrachD	0.09 (−0.18, 0.36)	0.50	−0.37 (−0.82, 0.09)	0.11	0.21 (−0.12, 0.53)	0.22	0.20 (−0.45, 0.85)	0.55
AIx	0.96 (−1.24, 3.16)	0.39	−0.54 (−4.15, 3.07)	0.77	−1.23 (−3.93, 1.48)	0.37	−2.67 (−7.96, 2.62)	0.32
PWV	0.08 (−0.06, 0.22)	0.26	−0.16 (−0.41, 0.10)	0.23	−0.06 (−0.24, 0.11)	0.47	−0.21 (−0.55, 0.14)	0.25

Each column represents an examination of a single exposure variable or study arm controlled for sex, race/ethnicity, caloric intake, physical activity, cotinine concentration and BMI category (except when the outcome examined was BMI);

*p<0.05;

** p<0.01

BMI: Body Mass Index; BMIz: BMI z score; logTrig: log-transformed triglycerides; logChol: log-transformed total cholesterol; logLDL: log-transformed LDL cholesterol; logHDL: log-transformed LDL cholesterol; BrachD: brachial artery distensibility; AIx: Augmentation Index; PWV: pulse wave velocity

PTSD: posttraumatic stress disorder; MDD: major depressive disorder; GAD: generalized anxiety disorder.

Table 3

Logistic Regression Analysis of Dichotomous Outcomes (Columns) Associated with Exposures (Rows) (N=402 subjects)

	Overweight		PTSD		Major Depressive Disorder		Generalized anxiety disorder	
Exposure	***OR (95% CI)***	***p***	***OR (95% CI)***	***p***	***OR (95% CI)***	***p***	***OR (95% CI)***	***p***
Home Dust	0.94 (0.86, 1.03)	0.20	1.07 (1.01, 1.14)	**0.03** *	0.91 (0.84, 0.10)	**0.04** *	1.03 (0.99, 1.08)	0.19
Dust Cloud	0.94 (0.84, 1.05)	0.26	1.12 (1.04, 1.21)	**0.004** **	0.92 (0.82, 1.02)	0.12	1.01 (0.96, 1.07)	0.58
Traumatic exposure	0.97 (0.89, 1.06)	0.50	1.07 (1.01, 1.13)	**0.04** *	1.01 (0.93, 1.09)	0.88	1.03 (0.99, 1.08)	0.14
WTCHR	0.90 (0.83, 0.98)	**0.01** *	1.10 (1.03, 1.16)	**0.002** **	0.94 (0.86, 1.02)	0.13	0.10 (0.96, 1.04)	0.88
PTSD	1.03 (0.90, 1.19)	0.64						
MDD	0.99 (0.90, 1.10)	0.90						
GAD	1.03 (0.84, 1.27)	0.76						

PTSD: posttraumatic stress disorder; MDD: major depressive disorder; GAD: generalized anxiety disorder.

* p<0.05;

** p<0.01